

Fall 2012 Study Guide for Plasma Membrane I and II (Dr. Smas) (One page):

1. What type of molecule underlies the basic architecture of the plasma membrane? Why are they called amphipathic? What type of molecular interactions underlie assembly and maintenance of the lipid bilayer? Why is the plasma membrane termed "fluid mosaic"? In what ways can integral or peripheral proteins associate with or embed in the plasma membrane? What about in the specific case of "glycophorin A"?
2. Why is the hydrophobic α -helix a highly suitable structure that allows transmembrane proteins to span the plasma membrane? What types of "R" groups/amino acids might you predict to be present or absent in this type of α -helix?
3. Cholesterol in the membrane leads to a diminution/abolishment of the phase transition. Think of it as having a "buffering" effect on membrane fluidity. What are the effects of increasing cholesterol content at: Above the T_m ? Below the T_m ? of an artificial simple membrane. How would membrane fluidity/ T_m be affected by alteration of the degree of bond saturation or the length of the fatty acyl chains?
4. If you wanted to "extract/remove" an integral membrane protein (like "glycophorin A") away from the lipid bilayer, what types of experimental conditions/treatments might you employ?
5. Give one example of how changes in the lipid composition of the extracellular and intracellular faces of the plasma membrane might signal cells for destruction, specifically the role of phosphatidylserine.
6. Using "glycophorin A" as an example, at what stage and where in the cell during protein synthesis are the sugars attached (glycosylation). Where on the protein do they occur (in general terms, which 3 types of amino acids)?
7. What types of substances are least likely to pass through the plasma membrane without a transporter protein? Give an example of a ligand-gated and a voltage-gated ion channel (as presented in class). What are their key structure/function features?
8. In the example of a "voltage gated K^+ " channel, the first 20 or so amino acids form a globular region referred to as "ball". How is this "ball" involved in function of this ion channel?
9. What is the function of the " Na^+/K^+ ATPase", at which point in the function do "cardiotonic steroid" drugs act, and with what result?
10. What is the underlying defect in "Myasthenia Gravis"? What model is proposed for the conformational change in this receptor that allows for controlled ion passage? What is the role of auto-antibodies in this disease?
11. In insulin-sensitive cells, such as a fat cell, what is the role and location of insulin, glucose, insulin receptor, and "GLUT4" in regard to the uptake of glucose into the fat cell? What type of transport mechanism is this (i.e. For insulin-stimulated glucose uptake)? What is meant by translocation of GLUT4 to the plasma membrane? Where does the insulin come from? Where is the insulin receptor located? As occurs during insulin stimulated glucose uptake, does either insulin or the insulin receptor actually enter the fat cell or does it function via "signal transduction" at the extracellular face of the plasma membrane? (HINT: THE ANSWER IS NO, Glucose enters through GLUT4, insulin stays outside and interacts with the extracellular region of the insulin receptor, in a form of "signal transduction")
12. Of the examples presented in detail in lecture, which types/classes of transporters are directly involved in binding and hydrolyzing ATP? Give two examples. How is one of these involved in drug resistance in cancer chemotherapy? What does the ABC stand for with this type of transporter? How is another (CFTR) involved in cystic fibrosis? What ion is transported by CFTR? What is the key distinction between passive and active transport mechanisms?
13. What types of interactions occur at an ion channel "selectivity filter"? In simple terms, for a K^+ channel, why is passage of K^+ energetically favored while passage of Na^+ is not? What is the role of the hydration shell?
14. How would an RBC with an intact cytoskeleton and plasma membrane respond to isotonic, hypertonic, or hypotonic saline (as covered in class)? What about a RBC from a patient with "hereditary spherocytosis"? Where is "glycophorin A" and "spectrins" in relation to the RBC plasma membrane